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Evolution of Human Growth Prolongation

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ABSTRACT This investigation evaluates hypotheses that seek to explain temporal retardation or prolongation of human ontogeny. Current hypotheses that address this issue are poorly defined and conflate several distinct theoretical positions. A model that predicts homogeneity in the extension of human growth periods is evaluated. This model is contrasted with two alternatives. The first alternative predicts heterogeneity in the extension of human growth periods. The second anticipates that human growth prolongation is the result of the uniquely derived "insertion" of a human childhood period into an ancestral ontogenetic trajectory. Allometric analyses of body mass growth data from 21 species of anthropoid primates suggest that human female and male ontogenies often depart from patterns established by other primates, but these departures are not uniformly exceptional. Comparisons imply that derived changes in human growth are heterogeneous. Relative to interspecific expectations, early growth periods are much prolonged, but later growth periods are actually reduced. Moreover, the attributes of early growth periods, including growth rates, timing of growth events, and size-for-age, are highly variable across primates. Low correlations among growth periods suggest independence among growth phases. These analyses highlight minimal distinctions between competing models (heterogeneous extension and insertion hypotheses) that attempt to explain human growth prolongation. More important, the present study facilitates refinements of causal models that have been proposed to explain human growth prolongation. Specifically, human growth prolongation may be related to derived changes in patterns of brain development. Alternatively, metabolic factors may have exerted influences on human ontogeny. However, models that predict long growth periods as a byproduct of metabolic factors do not adequately explain temporal retardation of human ontogeny. Am J Phys Anthropol 107:331–350, 1998.

A long period of growth and development clearly demarcates humans from other mammals. The unusually long duration of human ontogeny has been recognized and discussed by Western philosophers at least since the 1680s, while the biological implications of prolonged growth have been considered since the 1870s (Fiske, 1909; Lovejoy, 1959). More recently, Gould formalized this idea, emphasizing its relevance to the field of human evolutionary biology by arguing that:

> *"a general, temporal retardation of development has clearly characterized human evolution. This retardation has established a matrix within which all trends in the evolution of human morphology must be assessed"* (1977: 365; emphasis in the original).

Gould uses retardation to mean temporal delays in the initiation of various phases of growth. This concept of temporal retarda-

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tion provides insight into the evolution of human growth, given the long period between birth and maturation seen in humans when compared to other mammals and primates (Schultz, 1956). However, in this statement and elsewhere Gould uses retardation in reference to reductions in rates of shape change (development). Unfortunately, his variable usage of the term has resulted in confusion regarding the relations between the timing of growth events and rates of shape change (Godfrey and Sutherland, 1996). This theoretical disorder leaves numerous problems regarding the evolution of the timing of human growth unresolved. For example, Gould's notion of temporal retardation does not specify which of several potentially distinct human growth periods have undergone the greatest increases in duration. Gould does propose that the period of human gestation is relatively short (Gould, 1977; Montagu, 1961; Portmann, 1941, 1944; but see Martin and MacLarnon, 1990), but like previous researchers (e.g., Krogman, 1972), Gould does not specify the precise periods of postnatal ontogeny that have undergone the greatest evolutionary change. The clearest manifestation of this problem is evident in Schultz's famous diagram of primate life histories (1956: Fig. 57). This well-known figure, now a common element of introductory textbooks, illustrates protraction of all stages in human ontogeny (cf. Boaz and Almquist, 1997; Jurmain et al., 1997; see also Fleagle's (1988) insightful modification to this figure). Thus, the possibility that the human pattern of temporal retardation could have been produced through several distinct evolutionary pathways has not been thoroughly evaluated. Specifically, human growth prolongation may be attributable to uniform extensions in all growth periods present in an ancestral ontogeny, as implied by Schultz. In this case, the long period between birth and maturation may be the result of delays in the initiation of each phase of ontogeny. Alternatively, prolongation of human growth may stem from heterogeneity in patterns of temporal delays, with prolongation of a single growth phase resulting in an increase in the duration of the entire growth period. Lumping all phases of postnatal ontogeny into a single category spanning the period from birth to growth cessation neglects important variation in the duration of distinct growth periods. The patterning of temporal delays may have critical life history consequences, and can thus be traced to differing social and ecological selective forces (Bogin, 1988; Periera and Altmann, 1985; Leigh, 1995).

Hypotheses that specify temporal retardation of one or more phases of growth compete with alternatives that stipulate evolutionary "insertions" or additions of novel growth periods into ancestral ontogenetic trajectories. For example, Laird suggested that human growth is distinguished from growth in rhesus monkeys (*Macaca mulatta*) and "common" chimpanzees (*Pan troglodytes*) by:

> "the insertion [of] . . . a later preadolescent linear increase in body weight . . . connecting the linear component of infant growth with the period of rapidly increasing body weight that marks adolescence" (1967: 352).

She proposed that this period of growth was uniquely derived in humans. Gould (1977) responded to Laird's inference by questioning whether or not homology in patterns of ontogeny could be assessed solely on the basis of growth curves. His discussion of Laird's research indicates that he advocates general temporal retardation over insertion of developmental periods. Moreover, Gould's conceptualization of human heterochrony may require such a view (although there are major weaknesses in Gould's view of human heterochrony [Bogin, 1988, 1997; Shea, 1989]).

Bogin (1997) augments Laird's hypothesis by suggesting that childhood represents a human growth period not observed in other primate species. According to Bogin, the period of childhood is uniquely derived in humans, possibly appearing in *Homo erectus*, with *Homo sapiens* further extending this stage. Bogin defines the added childhood interval as "*the period following infancy, when the youngster is weaned from nursing but still depends on older people for feeding and protection*" (1997:64, emphasis in the original). This view suggests that prolonged human ontogeny is not a product of protracting growth periods seen in ances-

tral forms. It should also be noted that Bogin suggests that the human period of adolescence, as recognized by the adolescent growth spurt in skeletal length, is autapomorphic (uniquely derived) (Bogin, 1994, 1997; Bogin and Smith, 1996). While this does not hold for body mass growth, the situation for skeletal growth spurts in nonhuman primates remains unresolved (Leigh, 1996).

The lack of theoretical clarity regarding human growth prolongation compels further study of this phenomenon. Unfortunately, theoretical problems are compounded by limited empirical data. For example, earlier comparative studies included too few species for robust comparative allometric and phylogenetic analysis (e.g., Gavan and Swindler, 1966; Laird, 1967; Watts and Gavan, 1982). Consequently, inferences regarding the length of the human growth period lack a firm empirical foundation. While a derived shift towards a prolonged human growth period is a virtual certainty, inadequate comparative information precludes interpretative analyses. Additional research in this area requires a comprehensive comparative framework with attention to allometric and phylogenetic factors. Therefore, the initial objective of the study is to empirically assess various phases of ontogeny across primates, with special emphasis on the relation of humans to size-based expectations. Meeting this objective allows a determination of whether or not uniform delays in the timing of human mass growth are evident. In other words, the possibility that all periods of human ontogeny are extended is evaluated. The next objective is to consider the possibility that "inserted" periods of growth account for the long duration of human ontogeny. Specifically, if the period of childhood (Bogin, 1997; Laird, 1967) is inserted into human ontogeny, then the characteristics of early human body mass growth should be markedly distinct from other primates. Finally, this study attempts to gain insight into the evolutionary significance of human ontogeny by identifying causal factors that may explain the observed patterns. Recent research in developmental neuroscience, primate ontogeny, and paleoanthropology provide new insight into the evolutionary bases of human growth prolongation.

TABLE 1. Species evaluated by this study; selection of species is based on the presence of a subadult growth spurt (see Leigh, 1996)

Species	Sample size (M/F)	Growth spurt (by sex)	Plot identifier
Ceboidea (New World monkeys)			
Cebus apella	26/28	Male	a
Cercopithecoidea (Old World monkeys)			
Cercopithecus aethiops	30/30	Both	b
Cercopithecus mitis	27/37	Male	c
Cercopithecus neglectus	29/23	Male	d
Erythrocebus patas	41/52	Both	e
Cercocebus atys	38/71	Male	f
Macaca arctoides	52/58	Male	g
Macaca fascicularis	13/13	Both	h
Macaca fuscata	64/71	Both	i
Macaca mulatta	52/58	Both	j
Macaca nemestrina	39/64	Both	k
Macaca silenus	39/41	Male	l
Papio hamadryas	33/53	Male	m
Mandrillus sphinx	49/59	Both	n
Colobus guereza	46/49	Both	o
Presbytis entellus	29/24	Both	p
Presbytis obscura	19/17	Male	q
Hominoidea (apes)			
Gorilla gorilla	77/64	Both	r
Pan paniscus	13/23	Both	s
Pan troglodytes	22/23	Male	t
Homo sapiens	Literature data	Both	♀,♂

MATERIALS AND METHODS

Data for this analysis include body mass and age measures for 21 species of primates (Table 1). Observations are derived from captive animals held at zoological parks and primate centers throughout the world. All individuals are clinically normal and, with a handful of exceptions that were obviously adult size when weighed, exact ages are known for every individual. While the analysis of captive specimens may be less than ideal, the paucity of data from known-age wild populations precludes comparative analyses of absolute growth in either mass or skeletal length. It should be noted that virtually all of the data utilized in this study were obtained from group-housed (as opposed to single-caged) animals from rela-

tively large enclosures. Dietary parameters are comparable among species, limiting deviations that might result from short-term seasonal factors. Details regarding the composition of the sample are discussed elsewhere (Leigh, 1992a,b, 1994a,b; 1996; Leigh and Shea, 1995, 1996).

The set of species chosen for comparative analyses is derived from a larger sample of taxa (see Leigh, 1992a). The major criterion for selection is the presence of a body mass growth spurt in at least one sex (details on the basis for inclusion of taxa are presented in Leigh, 1996). Additional data regarding the sample, including bivariate plots of body mass growth rate curves, have also been presented previously (Leigh, 1996). Selecting only those species with subadult body mass growth spurts theoretically means that species with homologous patterns of mass growth are analyzed. Obviously, whether or not these growth curves include homologous features is not well established. Such a decision should be informed by investigations of hormonal mechanisms or genetic attributes, but information regarding these traits is currently unavailable. Data for interspecific analyses of skeletal ontogeny among primate species based on animals of known age are not presently available. In all, it should be recognized that results from this study have important limitations that require future investigation. Nevertheless, if a pervasive pattern of human growth prolongation is biologically important, then this pattern should be evident in comparative analyses of body mass ontogeny.

Human data are derived mainly from Buckler's analysis of adolescents in Leeds, England (1990). This source presents very detailed information regarding patterns of growth in this population. In addition, data for earlier growth periods are derived from standards published by Tanner et al. (1966). Given substantial variation in human growth throughout the world (Eveleth and Tanner, 1990), a broader comparative sample of humans would be optimal. However, data at the level of detail presented by Buckler and Tanner et al. are rarely available, even though mean-based estimates for many human populations have been published (Eveleth and Tanner, 1990). Unfortunately, extensive analyses of mean-based estimates provided highly variable measures of growth rates at peak velocity. Variation in peak velocity estimates resulted from small sample sizes, cross-sectional treatment of data, and aggregation of data into yearly age intervals. Evaluations of comparative human data show that estimates of all other variables were consistent across samples, with limited size-related variation. Thus, the British sources adequately characterize the human species for the purposes of interspecific comparisons. It can also be noted that the British data are derived from populations under relatively high quality nutritional conditions, which is also true of the captive nonhuman primate data utilized in this study.

Growth curve analysis

Ontogenetic data were analyzed at several different levels. First, regression analyses of growth in time for each sex in each species was undertaken (see Leigh, 1992a,b). These analyses treat data cross-sectionally, which eliminates the opportunity to analyze individual variation in growth (Tanner, 1978). The possible limitations of cross-sectional analysis have been discussed in detail with regard to this dataset (Leigh, 1996). Cross-sectional data appear to provide more accurate estimates of growth parameters in nonhuman primates than in humans. Essentially, the shorter growth periods of nonhuman primates reduces the distortion of peak velocities that often characterizes human cross-sectional data.

The next step in the analysis focuses on calculation of growth rate or velocity curves. Velocity curves provide the basic data for allometric components of the study. Velocity curves for each of the species analyzed in the present study have been published previously (Leigh, 1996), and protocols followed by this study are identical. These published velocity curves should be consulted in order to compare ontogeny qualitatively across species. Velocity curves were calculated by "double-smoothing" of the data with lowess regressions (Cleveland, 1979; Cleveland and Devlin, 1988; Efron and Tibshirani, 1991) followed by spline regressions (Schluter, 1988). These regression techniques free re-

searchers from the assumption that a single growth model adequately describes growth in all species. Specifically, lowess regressions provide an empirical representation of growth curves, but do not provide an equation from which a first derivative (rate of change or velocity) can be calculated. Consequently, a "pseudo-velocity" measure is derived by calculating the difference in successive predicted mass (Y) values, and dividing this difference by the difference in successive age (X) values. Lowess provides, in some cases, highly localized fits to data that sometimes exaggerate the rate of change. Consequently, lowess predicted values were subjected to a second round of smoothing with spline regression (Schluter, 1988). Splines describe the bivariate distribution using a more global fit on the data than lowess regression, effectively smoothing the irregular peaks implied by lowess smooths. Values output from the spline regressions are then differenced in order to calibrate the rate of growth. This approach provides fits that are very comparable to fits derived from parametric models (Leigh and Shea, 1996) and are consistent with longitudinal data. Thus, despite the complexity of the procedures this protocol appears to yield an accurate representation of growth among species.

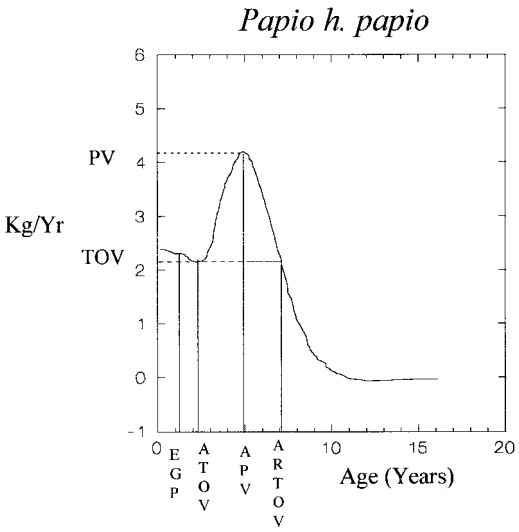

Fig. 1. Attributes of pseudo-velocity curves measured in the present study. The X axis represents age (in years), and the Y axis represents pseudo-velocity estimates (kg/yr) for male red baboons. Dashed lines represent velocity measures: PV = peak velocity, TOV = takeoff velocity. Solid lines show measures of age: EGP = early growth point (one-half age at takeoff velocity), ATOV = age at takeoff velocity, APV = age at peak velocity, ARTOV = age at return to takeoff velocity. Growth spurt duration is measured by subtracting age at takeoff velocity from age at return to takeoff velocity. Measures of size are represented by average adult mass. Early and late growth spurt duration are calculated by subtracting ATOV from APV, and APV from ARTOV, respectively.

Variable definitions

Data for allometric analyses are extracted from growth rate curves for each sex within species. These measures provide a comparative understanding of metric attributes of growth across species. This procedure focuses on attributes of growth spurts to define variables for analysis (Fig. 1). These measures are defined geometrically. *Peak velocity* is defined as the maximum observed postnatal velocity. *Age at peak velocity* is defined by dropping a line from peak velocity that is perpendicular to the "age" axis. The next variable, *takeoff velocity*, is estimated less precisely, and is defined by an inflection point that marks the beginning of the subadult body mass growth spurt. Typically, this variable is readily apparent, even in growth data for human populations described by mean mass data. As with peak velocity, the *age at takeoff velocity* is recorded by a line perpendicular to the "age" axis. *Age at return to takeoff velocity* is defined by the intersection of the line calibrating takeoff velocity and the declining rate curve. Age at return to takeoff velocity is calculated by a perpendicular from this line to the age axis. The *early growth point* is defined by the age at one-half the age at takeoff velocity. Variables estimated at this point in time provide a basis for comparing the human childhood period with other species. The *early growth rate* is derived by regressing body mass against age for the interval from birth to age at takeoff velocity with quadratic regression. The first derivative of this equation at the early growth point provides the estimate of early growth rate utilized in the study. This procedure was necessary because the spline regressions may not return predicted values for the age at the early growth point. All esti-

mates were compared with estimates derived from nonparametric smoothing, and were very similar.

Size at various points during ontogeny was analyzed. Neonatal mass data are provided from literature sources, including data collected for the present study (see Smith and Leigh, 1998). Mean male mass and female mass at the age of: the early growth point, takeoff, and peak velocity are analyzed. These data provide an indication of relative size across the sample.

Growth spurt duration is calculated by subtracting age at takeoff velocity from age at return to takeoff velocity. Thus, *early growth spurt duration* is estimated by subtracting age at takeoff velocity from age at peak velocity, and *late growth spurt duration* is calculated by subtracting age at peak velocity from age at return to takeoff velocity.

Allometric analyses examine the relations between all of these variables and species size, which is defined by the natural logarithm of average adult (asymptotic) mass (Leigh, 1992a,b). Allometric regressions are divided into categories according to the type of variable analyzed. 1) Regressions of velocities (early growth rate, takeoff velocity, peak velocity) provide measures of relative growth rates. These measures are assessed against both adult size and against average size-for-age. For example, early growth rate is regressed on adult size, then on average size at the early growth point. 2) Regressions of mass at various stages (neonatal, early growth point, takeoff point, and peak velocity point) against adult size give an indication of relative size-for-age. 3) Regressions of timing variables (age at takeoff, age at peak velocity, age at return to takeoff velocity, growth spurt duration, early and late growth spurt durations) against adult mass measure the relative pace of ontogeny. All regressions are reduced major axis fits calculated through Systat statistical software (Wilkinson et al., 1992).

Humans are excluded from calculation of regression lines and equations so that the departure of humans (by sex) from size-based expectations can be assessed. Values for humans are superimposed on regressions derived from the comparative sample. Inferences about the position of humans relative to the comparative sample are based on 95% confidence intervals for a predicted Y value given an X value (Sokal and Rohlf, 1981). Thus, the positions of human residuals are evaluated relative to the likelihood that the observed values represent predicted values. Male and female regressions are calculated separately in all cases. Letters identifying each species in plots are presented in Table 1. Phylogenetic adjustment of data follows procedures presented by Garland and Adolf (1994) and Felsenstein (1985). Correlations of phylogenetically adjusted data are determined by regression through the origin, as recommended by Garland and Adolf (1994).

RESULTS

Velocity measures

Velocity and adult size. Measures of velocities across primates generally show positive, but quite variable, correlations with adult size (Table 2). Beginning with the earliest velocity measure and moving to the latest, humans exhibit slow growth at the early growth point (midway between birth and the initiation of the subadult growth spurt) (Fig. 2). Human values are within the range of residual variation defined by other species, but outside the 95% confidence intervals for predicted values. The male human value is closer to the confidence interval than the female value. The scaling relation also suggests that early growth rates for females are relatively higher than comparable growth rates for males.

Takeoff velocities show a similar set of scaling patterns (Fig. 3). Humans present negative residual values when takeoff velocity is regressed against adult size, but are well within the range of variation defined by primates. The female human value is within the confidence interval, but the male human value is slightly outside the interval. As with early growth rates, females are expected to grow relatively faster than males of similar size throughout the range of sizes observed in this sample. Despite relatively higher growth rates, females regularly grow absolutely slower than males of the same species at this stage of growth. Thus, sexual dimor-

TABLE 2. Regression statistics for relations described in Figures 2–17; confidence intervals refer only to slope values; regressions for age at takeoff velocity are not calculated (see Fig. 12)

Variable pair	Intercept	Slope	Lower CI	Upper CI	Pearson product-moment correlation	Phylogenetically adjusted correlation
Male velocity at early growth point by adult mass	−1.47	0.71	0.56	0.86	0.90	0.91
Female velocity at early growth point by adult mass	−1.12	0.70	0.46	0.94	0.85	0.74
Male takeoff velocity by adult mass	−1.60	0.73	0.60	0.87	0.93	0.71
Female takeoff velocity by adult mass	−1.30	0.72	0.54	0.90	0.94	0.81
Male peak velocity by adult mass	−1.53	0.92	0.79	1.04	0.97	0.87
Female peak velocity by adult mass	−1.45	0.87	0.70	1.05	0.97	0.85
Male velocity at early growth point by size-for-age	−0.41	0.96	0.50	1.43	0.53	0.58
Female velocity at early growth point by size-for-age	−0.36	1.09	0.63	1.55	0.65	0.18
Male takeoff velocity by size-for-age	−0.85	0.91	0.54	1.28	0.70	0.86
Female takeoff velocity by size-for-age	−0.94	1.03	0.56	1.08	0.80	0.70
Male peak velocity by size-for-age	−0.96	0.89	0.59	1.07	0.95	0.68
Female peak velocity by size-for-age	−0.80	0.84	0.74	1.04	0.91	0.63
Male neonatal mass by adult mass	−2.51	0.71	0.57	0.85	0.94	0.32
Female neonatal mass by adult mass	−2.22	0.70	0.60	0.81	0.96	0.75
Male mass at early growth point by adult mass	−1.11	0.74	0.47	1.01	0.78	0.73
Female mass at early growth point by adult mass	−0.69	0.64	0.42	0.86	0.89	0.74
Male mass at takeoff by adult mass	−0.83	0.79	0.52	1.06	0.81	0.74
Female mass at takeoff by adult mass	−0.37	0.70	0.50	0.91	0.90	0.81
Male mass at peak velocity by adult mass	−0.64	0.92	0.79	1.04	0.97	0.98
Female mass at peak velocity by adult mass	−0.64	1.03	0.94	1.11	0.97	0.95
Male age at peak velocity by adult mass	0.15	0.46	0.28	0.65	0.72	0.72
Female age at peak velocity by adult mass	−0.64	0.57	0.16	0.98	0.63	0.43
Male age at return to takeoff velocity	0.71	0.40	0.24	0.55	0.74	0.74
Female age at return to takeoff velocity	0.11	0.52	0.22	0.82	0.76	0.79
Male early growth spurt duration by adult mass	−0.38	0.45	0.33	0.57	0.85	0.96*
Female early growth spurt duration by adult mass	−1.47	0.72	0.38	1.07	0.71	0.61
Male late growth spurt duration by adult mass	−0.60	0.43	0.20	0.64	0.49	0.79*
Female late growth spurt duration by adult mass	−1.90	0.79	0.36	1.23	0.73	0.81
Male total growth spurt duration by adult mass	0.31	0.41	0.27	0.55	0.77	0.77
Female total growth spurt duration by adult mass	−0.81	0.70	0.37	1.02	0.76	0.95

* These correlations are strongly affected by the contrast between *Presbytis (Semnopithecus) entellus* (Hanuman langur) and *Presbytis obscurus* (spectacled langur). The correlations drop markedly because of an apparently derived reduction in the duration of the growth spurt in *P. obscurus*. The correlation for early growth spurt and size drops to .59, while the adjusted correlation for the later growth spurt period drops to 0.40.

phism in adult size is reflected by this regression.

Peak velocity is tightly correlated with adult size (Fig. 4). Male and female reduced major axis regression lines overlap throughout the range of the data, and humans are very close to interspecific expectations. Humans are within their respective confidence intervals. Male and female regression lines overlap, and correlations are much higher than in regressions for velocities at earlier ages (Figs. 2, 3). Variation in the middle portion of the data scatter is quite high. Variation among hominoids is limited.

In general, human growth rates are quite consistent with interspecific expectations. While human growth rates relative to adult size tend to be low, they are typically within the range of residual variation. For peak velocity, human estimates are well within confidence intervals for predicted values, and match interspecific expectations almost exactly. Differences between sexes in scaling relations are apparent in plots representing earlier growth periods.

Velocity and size-for-age. Plots of early growth rates scaled against size-for-age present a wide range of correlations. Regression of growth rates at the early growth point show moderate correlations, particularly for the male scaling relation for unadjusted data and a very low correlation for the female adjusted correlation (Fig. 5, Table 2). Humans are outside the confidence intervals for this regression, with negative residual values. The low correlation reflects substantial variation among primates in the determinants of size during early growth. This relation reflects variance in neonatal

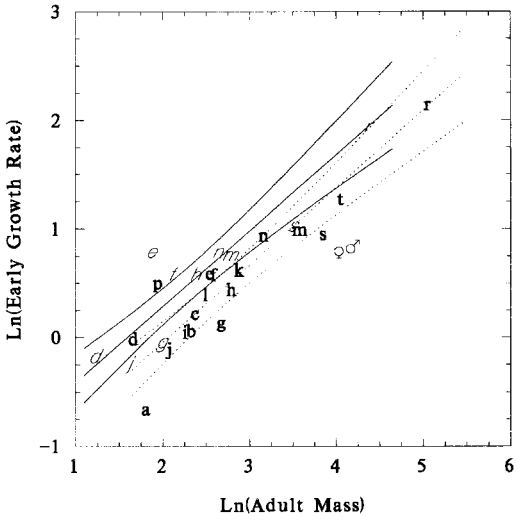

Fig. 2. Reduced major axis regression of growth rate at the early growth point (kg/yr) against adult mass (kg). Note A: Letters designate each species with italic fonts indicating female datapoints and bold fonts representing male datapoints (see Table 1 for key); solid and dotted lines represent female and male regressions, respectively; confidence intervals for predicted values are shown for each regression; human estimates are shown by symbols for each sex. Note B: Human values are superimposed on the primate-wide regression.

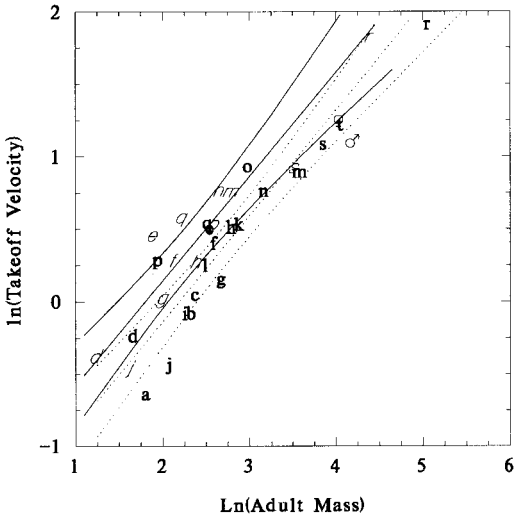

Fig. 3. Reduced major axis regression of takeoff velocity (kg/yr) against adult mass (kg). See Notes A and B, Figure 2.

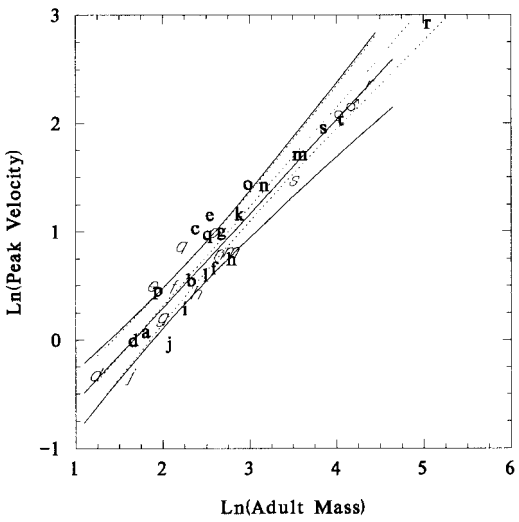

Fig. 4. Reduced major axis regression of peak velocity (kg/yr) against adult mass (kg). See Notes A and B, Figure 2.

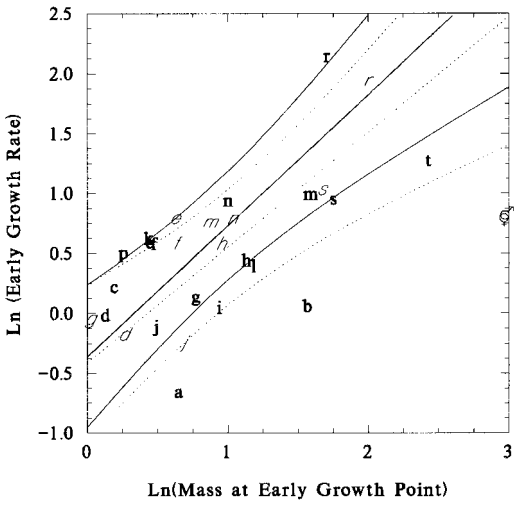

Fig. 5. Reduced major axis regression of velocity at the early growth point (kg/yr) against size-for-age (kg). See Notes A and B, Figure 2.

mass, which affects the size axis, as well as variation in postnatal growth rates. Moreover, the reduced major axis regression technique yields a high slope value when correlations are low. Thus, the human values are much closer to a least-squares line. In any case, the correlation between these variables is remarkably low, complicating estimates of size-based expectations. Considerable non-size-related variation exists between these variables.

Regressions of takeoff velocity against size-for-age also yield low correlations, especially for males (Fig. 6; Table 2). However, takeoff

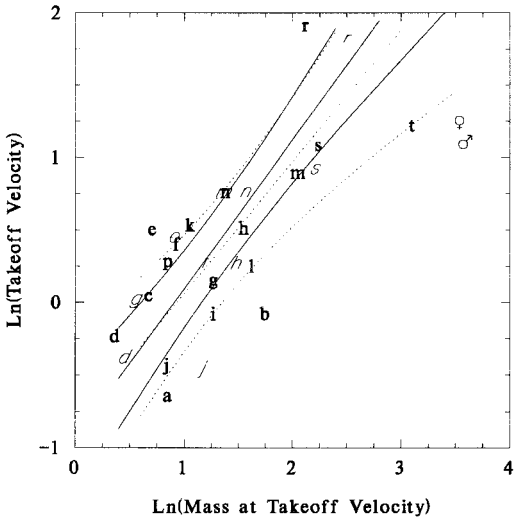

Fig. 6. Reduced major axis regression of takeoff velocity (kg/yr) against size-for-age (kg). See Notes A and B, Figure 2.

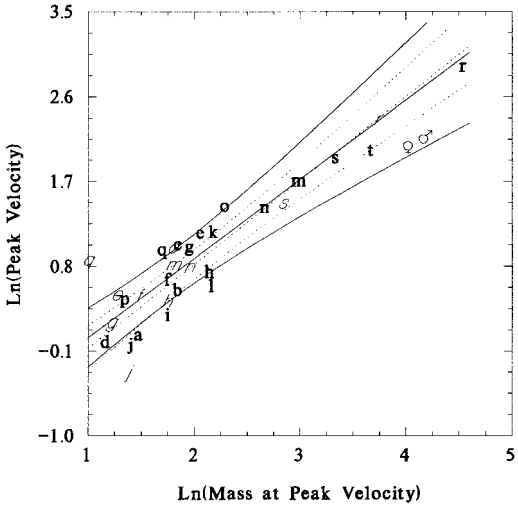

Fig. 7. Reduced major axis regression of peak velocity (kg/yr) against size-for-age (kg). See Notes A and B, Figure 2.

velocity correlations are higher than those observed for the early growth rate comparisons, suggesting that variation in neonatal size is less of a factor in these regressions. Humans are outside the confidence intervals, again with negative residual values. Human males have slightly lower takeoff velocities relative to size-for-age than females. This pattern is consistent with the pattern implied by the primate-wide regression line. Much of the variation in these regressions is unrelated to size.

Peak velocity and size-for-age are tightly correlated (Fig. 7). As with previous comparisons of growth rates against size-for-age, human residual values are negative. However, these values are clearly within the range of interspecific variation, and the human female estimate lies within the confidence intervals. Like other size-for-age comparisons, regression lines for each sex overlap to a substantial degree.

Regressions of growth rates against size-for-age generally suggest that humans grow slowly compared to size-for-age. Correlations across species are low during the early growth periods, but increase as adult size is reached. Variance at early growth periods can be attributed partly to variance in patterns of prenatal growth and resulting differences in neonatal mass.

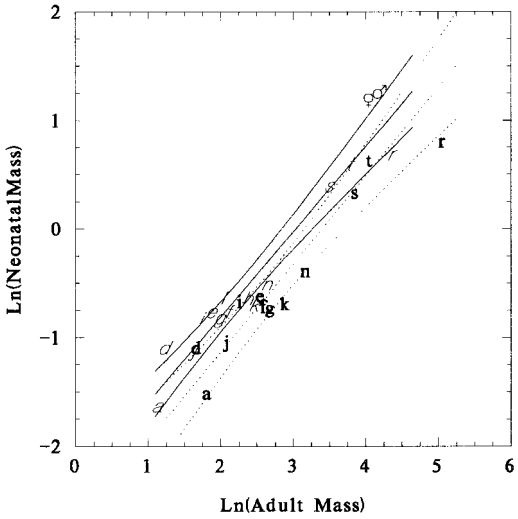

Fig. 8. Reduced major axis regression of relative size at birth (neonatal mass (kg) against adult mass (kg)). See Notes A and B, Figure 2.

Relative size

Comparisons of relative size complement assessments of velocities at size-for-age by describing size variation with age. Regression of neonatal mass against adult mass suggests that humans are relatively large at birth (Fig. 8). Both sexes are outside confidence intervals. The female regression line is transposed above that of the male line, suggesting that, across primates, females

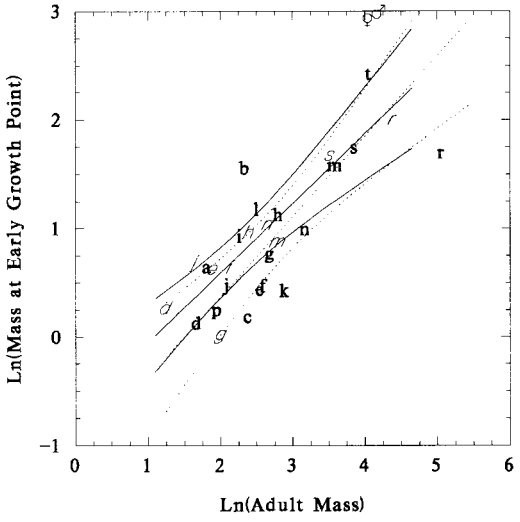

Fig. 9. Reduced major axis regression of relative size at the early growth point (mass at early growth point (kg) against adult mass (kg)). See Notes A and B, Figure 2.

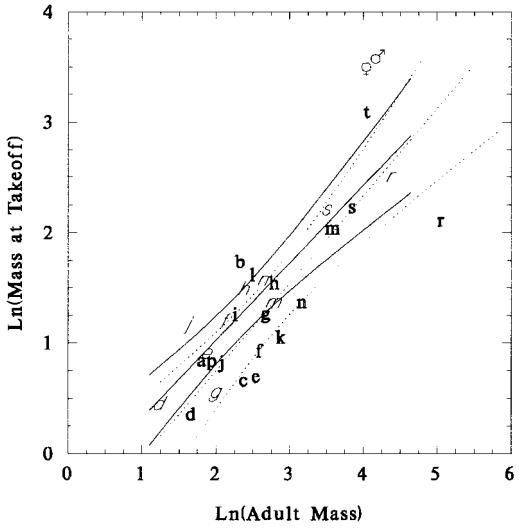

Fig. 10. Reduced major axis regression of relative size at takeoff velocity (mass at takeoff velocity (kg) against adult mass (kg)). See Notes A and B, Figure 2.

are born at a higher proportion of adult mass than males.

Size at the early growth point shows less relative sexual dimorphism than observed in the neonatal mass regression (Fig. 9). Consequently, both males and females are roughly similar in terms of proportion of adult size at this point in ontogeny. The human values appear as positive residuals that are outside the confidence intervals. A large degree of variation characterizes the relation across species.

At takeoff velocity, human sizes again appear as positive residuals. The human residuals represent extremes, and are outside the confidence intervals (Fig. 10). An extremely high degree of variation is present among hominoids. Gorillas (*Gorilla gorilla*, point "r") tend to be quite small at takeoff velocity, but chimpanzees (*Pan troglodytes*, point "t") and humans are large.

Human sizes are very close to the upper confidence limits at the age of peak velocity (Fig. 11). As with previous regressions, male and female regression lines overlap considerably. Mass at peak velocity is often close in age to the age at which adult size is reached, partly accounting for the high correlation. However, substantial residual variation is present in lower portions of the size range, which may simply reflect larger samples in this size range.

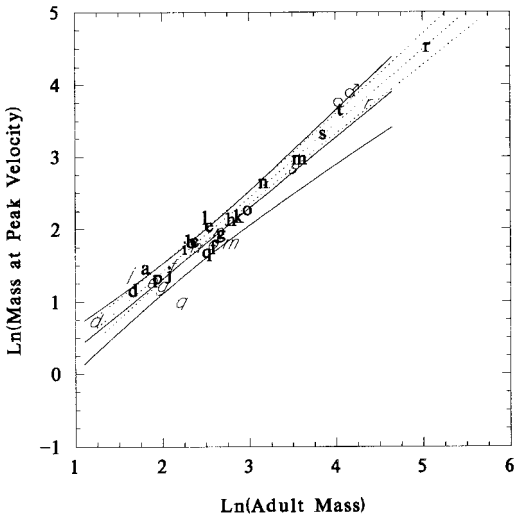

Fig. 11. Reduced major axis regression of relative size at peak velocity (mass at peak velocity (kg) against adult mass (kg)). See Notes A and B, Figure 2.

Overall, humans tend to be large at each point during growth relative to interspecific expectations. Large human size is especially evident in neonatal mass. The effects of large neonatal mass are carried through to later growth periods. Human values are

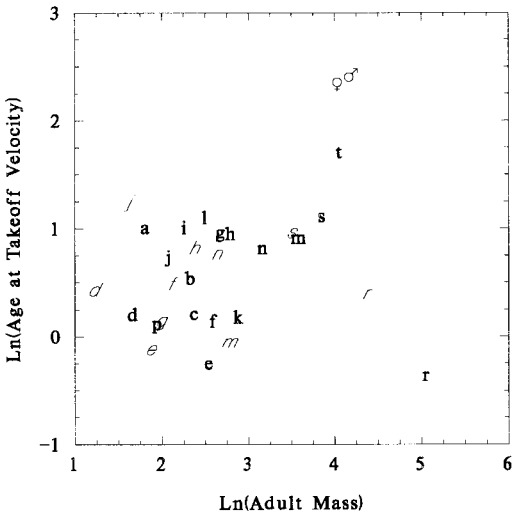

Fig. 12. Reduced major axis regression of age at takeoff velocity (years) against adult mass (kg). See Notes A and B, Figure 2.

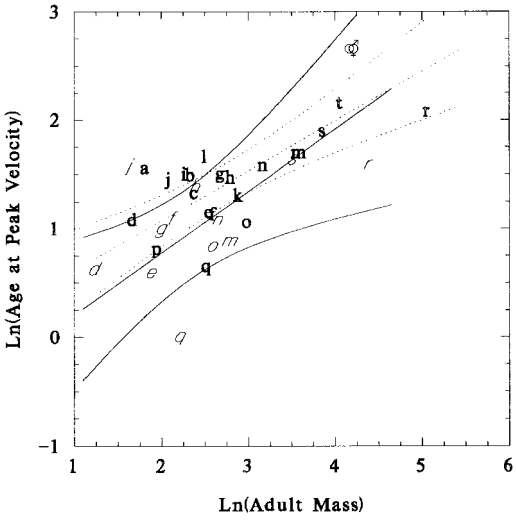

Fig. 13. Reduced major axis regression of age at peak velocity (years) against adult mass (kg). See Notes A and B, Figure 2.

roughly consistent with interspecific expectations later in the growth period.

Age measures

Scaling analyses. Age at takeoff velocity varies independently of size across primates (Fig. 12). Humans are quite distinctive in showing very late absolute ages at takeoff velocity. It can be noted that hominoids present the greatest range of variation in this variable relative to other taxonomic groups. Specifically, human takeoff age is quite late, but takeoff age for male gorillas is extremely early (see Leigh and Shea, 1995, 1996). Male "common" chimpanzee takeoff age is quite late, but pygmy chimpanzee (*Pan paniscus*) takeoff ages are about average for primates. Variation in takeoff age is also extreme within other groups of closely related species.

Age at peak velocity is positively correlated with size, but the correlation is not strong (Fig. 13). Male and female regression lines converge toward the upper size ranges. Humans present positive residuals and are within the confidence intervals based on other species. Human estimates are quite close to interspecific expectations for this variable, given the large absolute value in age at takeoff velocity for humans.

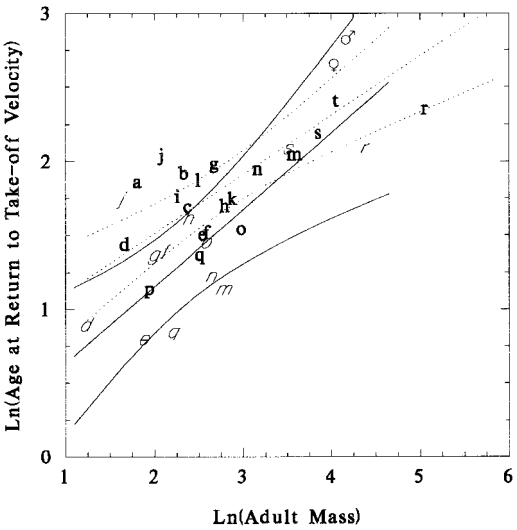

Fig. 14. Reduced major axis regression of age at return to takeoff velocity (years) against adult mass (kg). See Notes A and B, Figure 2.

Plots for age at return to takeoff velocity show that humans reach return to takeoff velocity rather late (Fig. 14). The human female estimate is within the confidence intervals.

All components of human growth spurts (early growth spurt duration, late growth spurt duration, and total growth spurt dura-

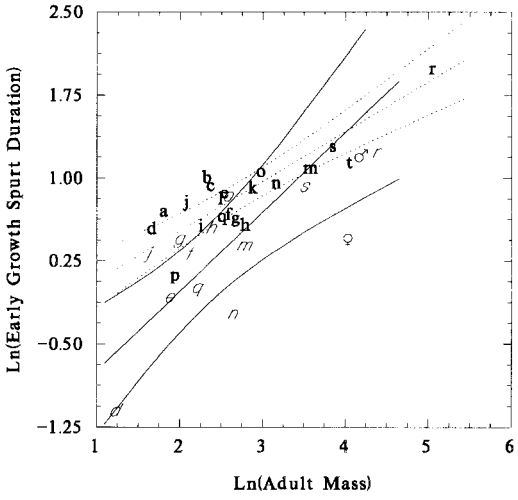

Fig. 15. Reduced major axis regression of early growth spurt duration (years) against adult mass (kg). See Notes A and B, Figure 2.

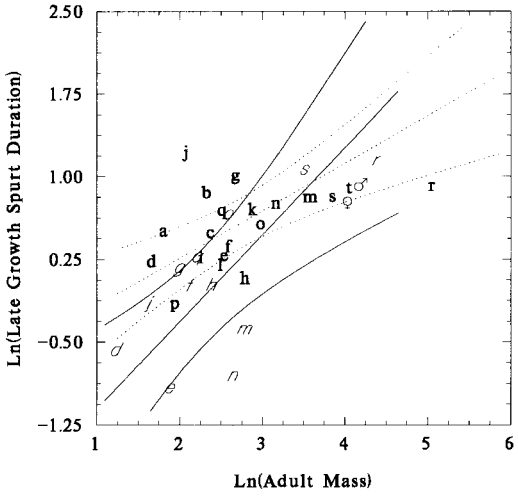

Fig. 16. Reduced major axis regression of late growth spurt duration (years) against adult mass (kg). See Notes A and B, Figure 2.

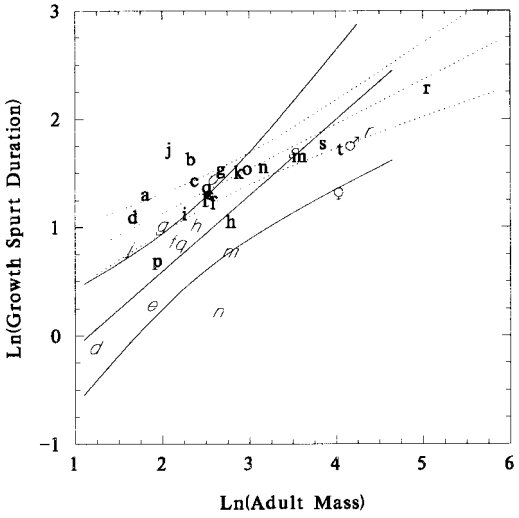

Fig. 17. Reduced major axis regression of total growth spurt duration (years) against adult mass (kg). See Notes A and B, Figure 2.

tion) are relatively short (Figs. 15, 16, 17). These regressions show positive correlations with size. Across primates, female values are much more variable than male values, and the scaling relations between the sexes converge in the upper size ranges. The first segment of the human male growth spurt is closer to a size-based expectation than the comparable estimate for human females. Late growth spurt duration presents a scaling pattern that is similar to previous graphs, with convergence in the upper size ranges (Fig. 16). Human values are well within the confidence intervals, but correlations for this variable are fairly low. Total growth spurt duration (Fig. 17) suggests that humans have comparatively short body mass growth spurts. The negative residuals for humans are slightly outside the confidence intervals.

In summary, measures of growth timing show extensive variation across primates, with no predictable relation between adult size and age at takeoff velocity. Measures for later points in time show stronger size correlations, which are consequences of moderately predictable relations between growth spurt duration and size.

Correlations among growth periods. Correlations for the variables age at takeoff velocity, early growth spurt duration, and late growth spurt duration are very low for both males and females, suggesting that the length of different growth periods varies independently across species (Table 3). This result is implied by earlier scaling results, which show that some timing variables are independent of size (e.g., age at takeoff velocity) while others are moderately to strongly correlated with size. Low correlations among these growth periods may re-

TABLE 3. Correlations among growth periods for males and females

	Age at takeoff M, F	Early growth spurt duration M, F
Early growth spurt duration	0.07, 0.13	—, —
Late growth spurt duration	0.27, 0.25	0.43, 0.86

flect the influence of separate factors on the duration of each period of growth. Despite generally low correlations, early and late growth spurt durations covary closely in females.

DISCUSSION

Mechanisms of human growth prolongation

Body mass ontogeny. This study confirms previous research suggesting that human growth is quite prolonged in comparison to other primates. Specifically, human body mass growth trajectories are very unusual when contrasted with empirical size-based expectations. Human growth is much longer than expected based on size mainly because of an extremely long early growth period. Later growth phases are reduced in humans relative to expectations, and thus do not contribute to human growth prolongation. Humans are "oversized" during early periods of growth, but at later stages female and male human body sizes approximate interspecific expectations. Substantial variation unrelated to size characterizes earlier segments of primate growth, but variation is reduced in later phases. Growth phases also appear to be statistically independent of each other. Thus, human growth prolongation results from derived changes during early postnatal growth periods rather than from uniform extension of all growth periods.

Analyses of body mass growth do not unambiguously resolve the issue of whether or not the period of human childhood is inserted into an otherwise ancestral trajectory (Bogin, 1997; Laird, 1967). Substantial residual variation in all aspects of early growth periods and uncertainty regarding an ancestral condition for humans complicate this assessment. The most important and greatest quantity of ontogenetic variation among primates occurs in the age at takeoff velocity, reflecting considerable size-independent variation in the duration of early growth periods. Humans exhibit the most extreme positive value for age at takeoff velocity, suggesting that this phase has seen the greatest prolongation over the course of human evolution. Variation across primates may indicate that evolutionary changes in the duration of this growth period can evolve quite readily (or at least independently of any theoretical associations with body size). Thus, the attributes of early growth periods appear to be highly evolvable (Houle, 1992) or responsive to selective pressures. If developmental timing and rates of body mass growth are so sensitive to selective pressure, then the human pattern of a long period of growth prior to takeoff velocity may have evolved readily from an ancestral condition in which this period is either quite short or not apparent. Both unadjusted and phylogenetically adjusted correlations between size and other variables (growth rates, relative growth rates, and relative size) increase with age, possibly suggesting that later periods are less susceptible to size-independent evolutionary change than early periods. Overall, appreciable size-independent variation is evident in early growth periods, blurring attempts to distinguish between "insertion" and "prolongation" hypotheses.

It is important to emphasize that humans show a qualitative pattern of ontogeny that is very similar to chimpanzees and gorillas. This observation may favor a "prolongation" hypothesis. For example, the general shape of body mass growth curves in these species (with the exception of female "common" chimpanzees) is very similar (Leigh, 1996; Leigh and Shea, 1995, 1996). Transformation of a male chimpanzee body mass growth curve to a human curve appears to be relatively straightforward (Rice, 1997). Despite the qualitative similarities among hominoids, these species are highly variable in terms of age at takeoff velocity, with high human values contrasting with exceptionally low values for gorillas. Unfortunately, this diversity complicates attempts to define an ances-

tral condition for humans, but it also strengthens the notion that the quantitative attributes of ontogenetic trajectories can respond rapidly to selective pressures.

Each type of variable (velocity, relative size, and timing) differs in patterns of correlations with size across this comparative sample. The lack of covariation among these different variables further indicates that the attributes of body mass growth periods are evolutionarily labile. For example, timing variables show weaker correlations with size than other kinds of variables at each point in ontogeny. Scaling relations for timing variables also tend to vary by sex to a greater degree than other variable types. Thus, the timing of growth events may evolve more readily independent of size than other variables. Such circumstances may be conducive to the evolution of extremes in the length of growth phases, such as seen in humans.

Skeletal, hormonal, and behavioral ontogeny. The insertion and prolongation hypotheses can be assessed by other lines of evidence. Specifically, comparative data regarding mid-growth spurts, skeletal growth spurts at puberty, adrenarche, and behavioral data are critical to resolving these issues. The "mid-growth spurt" in skeletal length has been identified as an "important feature of human childhood" (Bogin, 1997: 73), while growth spurts in stature at puberty in each sex are suggested as uniquely derived human features (Bogin, 1988). Several nonhuman primates exhibit mass growth spurts that appear to parallel the human mid-growth spurt (*Cebus apella* (capuchin monkey), *Macaca fuscata* (Japanese macaque), *Pan paniscus* (pygmy chimpanzee), and *Gorilla gorilla* (gorilla) (Leigh, 1996)). Mass data are not currently adequate to characterize these spurts with much accuracy, and appropriate skeletal growth data are simply not available. Similar problems preclude inferences about the evolution of human skeletal growth at puberty. Some studies find pubertal growth spurts in selected skeletal dimensions (Gavan (1982) and Watts and Gavan (1982) for chimpanzees (*Pan troglodytes)*; Tanner et al. (1990) for rhesus (*Macaca mulatta*); Crawford et al. (1997) and Leigh (1998) for baboons (*Papio hamadryas*)). Other studies fail to observe skeletal growth spurts (Cheverud et al., 1992, for toque macaques (*Macaca sinica*); see Bogin, 1988).

Hormonal data conceivably provide the most direct insight into this issue. For example, adrenarche, or increased secretion of androgens by the adrenal glands (Papadimas, 1997) has been cited as a diagnostic of human childhood (Bogin, 1997). Adrenarche seems to occur in chimpanzees (Cutler et al., 1978), but a marker of adrenarche, increased levels of dehydroepiandrosterone sulfate (DHEAS), is not apparent in baboons (Crawford et al., 1997). Data for other primates are unavailable, so the present study cannot address questions regarding this important system.

Behavioral data have been used to define various phases of ontogeny and can contribute to this debate (Bogin, 1997; Periera and Altmann, 1985). In fact, Bogin's argument for insertion of childhood into an otherwise ancestral growth trajectory relies partly on a behavioral definition of childhood. Specifically, he defines childhood as the post-weaning period during which offspring depend on older individuals for food. Rigorous evaluation of this definition requires investigation of behaviors across a broad comparative sample of primates. Pending such an analysis, it should be noted that this definition applies extremely well to tamarin and marmoset species (Garber, 1997). In these taxa, group members other than the mother actively participate in provisioning and protecting offspring for an extended period of time relative to total growth duration (Garber and Leigh, 1997). Long periods of offspring dependency are also characteristic of chimpanzees (Goodall, 1986). Consequently, reevaluation of behavioral definitions must be included in future investigations of this problem. Behavioral data do not support the view that post-weaning dependency is unique to humans.

Assessments of body mass ontogeny along with other lines of evidence indicate that temporal components of Gould's (1977) matrix of retardation is manifest through prolongation of early periods of human growth coupled with decreases in the duration of later growth phases. The unusual temporal

aspects of the human matrix of retardation are most evident early in postnatal ontogeny. In contrast, later human growth periods are brief relative to size-based expectations, and thus do not contribute to human growth prolongation. Consequently, a heterogeneous pattern of growth prolongation accounts for the human pattern more adequately than a proportional or uniform model of growth prolongation. It should be noted that insertions of novel growth periods remain a viable possibility that can be tested with additional ontogenetic data.

Evolutionary considerations

A number of factors can account for the evolution of a prolonged period of growth prior to the subadult growth spurt. Bogin (1997) thoroughly discusses these possibilities, which typically fall into two categories. The first category includes "traditional" explanations, which focus on learning and behavioral flexibility as causes of human growth retardation (Dobzhansky, 1962; Fiske, 1909; Poirier, 1977; Washburn and Hamburg, 1965). The second class of models emphasizes feeding adaptations as alternatives to learning models. Within this latter category, a metabolic risk aversion model has the most explicit implications for understanding variation in primate ontogeny (Janson and van Schaik, 1993). The present results have important consequences for understanding how well these models explain human growth prolongation, particularly because this study illustrates the importance of the early periods to human growth prolongation. In other words, models that explain the exceptional features of early segments of human ontogeny should have greater predictive power than models focusing on later ontogenetic periods.

Learning and behavioral flexibility. Most explanations of human growth prolongation assume that humans must assimilate large quantities of complex information prior to adulthood (Poirier, 1977; Tanner, 1978; Washburn and Hamburg, 1965). Enhancements of traditional learning models highlight the adaptive importance of human behavioral flexibility (Gould, 1977; Bogin, 1997). Unfortunately, traditional learning models are often diffuse, lacking explicit reference to mechanisms of learning and neural development. However, recent research in developmental neuropsychology provides an opportunity to refine our understanding of these issues. This research reveals at least two distinct phases of neural development in mammals, termed experience-expectant and experience-dependent periods (Greenough et al., 1987). The experience-expectant period involves, "the intrinsically governed generation of an excess of synaptic connections among neurons, with experiential input subsequently determining which of them survive" (Greenough et al., 1987:540). Essentially, this suggests that overproduction and subsequent pruning of excessive synaptic connections occurs in response to particular environmental stimuli during development of the brain. This process of forming primary sensory circuits requires taxon-specific stimuli that are predictably available during critical developmental periods. Exposure to abnormal stimuli (e.g., via strabismus of the eye) or deprivation of appropriate stimuli (e.g., dark-reared cats) can result in improperly organized neural systems (see Hubel and Wiesel, 1970). The subsequent experience-dependent period is responsible for "the storage of information that is unique to the individual" (Greenough et al., 1987:540). Events encoded along experience-dependent lines are more likely to be unpredictable and, therefore, less likely to occur during a prescribed critical developmental period. Experience-dependent information is encoded through either the formation of new synapses or modification of existing connections in response to newly acquired knowledge of the sensory environment (Kleim et al., 1997). Both the overproduction and subsequent loss of synaptic connections, as well as the modification and formation of synapses in response to external stimuli, are key components of brain development (Purves, 1988).

Prolongation of human growth, particularly through extension of the early growth periods, may reflect requirements imposed by the timing of human brain ontogeny relative to nonhuman primates. Prolongation of early growth periods may be especially critical because changes in synaptic

density in humans are not synchronized. Moreover, asynchrony in the development of brain regions seems to distinguish humans from nonhuman primates (Huttenlocher and Dabholkar, 1997). For example, synaptic elimination in the human auditory cortex (Heschl's gyrus) ceases by about 12 years of age, but persists in the prefrontal cortex (middle frontal gyrus) until mid-adolesence (Huttenlocher and Dabholkar, 1997). In contrast, synaptic elimination occurs concurrently among regions in rhesus monkeys (Rakic et al., 1986). Prolongation of human growth extends the amount of time available for these processes. Finally, human growth prolongation may ensure the retention of a functionally juvenile (i.e., highly plastic) brain for a long period of time. This promotes prolongation of experience-expectant and -dependent periods while enabling variability in the timing of brain ontogeny among regions. It may also promote comparatively high plasticity into the adult period.

Protracted and asynchronous experience-expectant and experience-dependent periods may underlie human adaptive flexibility. Rauschecker supports this proposal by suggesting that plasticity in the adult brain represents "the residue of cortical plasticity" seen in young animals (1995:7). More dramatic examples of human brain flexibility are provided by clinical studies. Cao et al. (1994) demonstrate the ability of the brain to reorganize after focal injury in young patients. Profound levels of reorganization are illustrated by studies of hemispherectomized patients (Vining et al., 1997). Vining et al. document the ability of younger hemispherectomized patients to reacquire language abilities that are within normal limits, even after removal of the left brain hemisphere (1997). Regional asynchrony in experience-expectant and -dependent phases of development conferred by temporal retardation (and possibly neoteny (Anton and Leigh, 1998)) may have played a major role in the evolution of these capabilities.

Recent paleoanthropological research provides insight into the mechanisms that selected for brain plasticity. Specifically, Potts (1996a) suggests that behavioral flexibility may be a hallmark of human evolution, ultimately arising as an evolutionary response to environmental variability (Bower, 1997; Potts, 1996a,b). The flexibility inferred by Potts may be intimately related to growth prolongation. Furthermore, the evolution of two key human traits may have established a foundation upon which pervasive delays in human neural development are based. Specifically, bipedalism probably required extensive modifications in the experience-expectant and -dependent periods involved in locomotion. One of the most pressing problems faced by bipeds is balance, which requires the integration of a number of neurological systems plus coordination among different anatomical systems (Woollacott and Shumway-Cook, 1989). For example, the development of balance occurs over a protracted period of time, with distinct stages observed at least until adulthood (Assaiante and Amblard, 1992; Woollacott et al., 1987). The component elements of bipedalism mature at different ages, and appear to be context-sensitive (Thelen and Ullrich, 1991). Electromyographic studies also suggest that patterns of muscle recruitment change with age, reaching an adult-like pattern by about 7 years of age (Okamoto and Kumamoto, 1972). Consequently, the development of bipedalism, especially of balance, undoubtedly required modification of existing neural circuits, impling changes in synaptic development. Temporal delays in ontogeny to enable "rewiring" may have been key components of the response of hominin ancestors to selection favoring bipedalism.

Language acquisition, which requires long periods of time, represents the second major adaptation that involved shifts in the nature and timing of experience-expectant and -dependent periods. Greenough et al. note that several brain components may be involved in language acquisition, each with "its own time course and experiential sensitivities" (1987:553). Clearly, humans are distinct from other primates in requiring a period of language acquisition. Temporal retardation could provide the mechanism through which high levels of experiential sensitivities to language acquisition are maintained well into postnatal life.

Human growth prolongation may be a necessary co-requisite of large human brain size (Finlay and Darlington, 1995) and the development of brain plasticity. Specifically, it can be hypothesized that delays in growth were necessary for the modifications in the experiential periods that ultimately enabled critical human adaptations such as bipedalism and language. This scenario implies that two major episodes of growth prolongation have occurred during the course of human evolution. Selection for bipedalism may have entailed changes in neural development that were facilitated by delays very early in postnatal ontogeny. These delays may have precipitated additional temporal delays associated with language acquisition, and may have been integral to the evolution of the human cognitive system.

Feeding adaptations. New research on the evolution of primate growth rates provides a compelling alternative to traditional learning models. Specifically, a metabolic risk aversion model predicts that extended primate growth periods may be a byproduct of adaptations to metabolic risks (Janson and van Schaik, 1993). According to this model, low primate growth rates are a result of a trade-off between predation and feeding competition. Predation favors group formation in primates, but increases costs of feeding competition, especially for juvenile animals. Under these circumstances, low primate growth rates serve as an adaptation to feeding competition within groups because slow growth minimizes metabolic costs per unit time. A consequence of selection against high growth rates may be that the total duration of ontogeny is extended.

Human growth rates relative to adult size are typically close to interspecific expectations. This finding implies that a postnatal risk aversion model may not apply with much force to humans. On the other hand, slow growth rates relative to size-for-age in humans may support a postnatal risk aversion model. However, variance in growth rates relative to size is difficult to interpret because of high variance in neonatal mass. Differences in neonatal mass reflect different patterns of prenatal investment (Martin and MacLarnon, 1990; Smith and Leigh, 1998). This investment appears to be fairly inexpensive for humans (Martin and MacLarnon, 1990).

Some consistencies with a postnatal metabolic risk aversion model seem to be evident for humans, but these stem from the requirements of brain ontogeny. The brain accounts for about 20–25% of energy consumed at resting metabolism in adults (Lehninger, 1982). More importantly, Holliday (1986) estimates that much higher percentages of energy are consumed by the brain in young individuals, with infants devoting 85% of energy at resting metabolism to the brain (Leonard and Robertson, 1992). Leonard and Robertson further suggest that humans are distinct from other primates in this regard (Leonard and Robertson, 1992, 1994; see also Aiello and Wheeler, 1994). Consequently, Leonard and Robertson state that human growth prolongation "may be partly an adaptation to limit the already high total brain energy requirements during childhood" (1992:191). Metabolic costs of the human brain are probably especially high during the time of absolute increase in brain size (prior to 6 postnatal years of age (Jolicoeur et al., 1988). As originally proposed, a metabolic risk aversion strategy might be expected to selectively favor slow brain growth over the entire course of ontogeny. The same adult size could result, but metabolic risks would be evenly distributed across the entire developmental period. Instead, the human strategy involves shifting these costs to the perinatal period. This strategy effectively distributes metabolic costs to the mother, another caretaker, or group of older individuals. Parallel strategies characterize some small-bodied New World monkeys (Garber and Leigh, 1997). Studies of alternative strategies for the distribution of metabolic costs may offer insight into human adaptations.

Concentration of metabolic costs during the perinatal period and deflection of these costs to older caretakers suggests that human growth prolongation cannot be attributed entirely to the consequences of metabolic risks encountered during postnatal ontogeny. Moreover, the large size of the human brain during this early period is mainly responsible for such risks. Selection

for prolonged and asynchronous experience-expectant and experience-dependent periods, which may require a relatively large and metabolically costly brain, can account for human growth prolongation.

CONCLUSIONS

A derived prolongation of human growth during early phases of ontogeny is consistent with comparative analyses of body mass growth trajectories across anthropoid primates. Thus, temporal aspects of Gould's (1977) matrix of retardation in human evolution involve changes that occur during periods of ontogeny prior to the initiation of the subadult growth spurt in mass. Prolongation of human ontogeny appears to be heterogeneous, with early periods of postnatal ontogeny lengthened relative to size-based expectations, but reductions in periods of growth during the subadult growth spurt. It should be emphasized that additional analyses of hormonal, skeletal, and behavioral ontogeny are needed in order to further understand this problem.

The attributes of early growth periods appear to be quite variable across primates when judged against body size. Velocities are the most predictable in relation to body size, but relative size, velocity relative to size-for-age, and estimates of the pace of ontogeny (timing variables) show appreciable amounts of variation independent of adult size. Variation that is unrelated to adult size is typical during early periods of primate ontogeny, and may reflect a high degree of adaptive variation. Low phylogenetically adjusted correlations for some of these variables suggest that attributes of early growth periods and adult body size are often uncoupled, and evolve independently.

The evolutionary causes of prolonged human ontogeny are most readily interpreted as requirements of the developing brain. Specifically, temporal retardation of human ontogeny may extend experience-expectant and experience-dependent phases of neural development for a number of different brain regions. Moreover, temporal delays of ontogeny enable independence among brain regions in the time course of these phases. Key human adaptations that can be linked to prolonged human ontogeny include bipedalism and language. An important co-requisite for these adaptations includes a prolonged period of ontogeny, facilitating new patterns of neural development. These results are consistent both with new clinical evidence on brain plasticity and with paleoanthropological data suggesting an impact of environmental variation on the evolution of brain plasticity. Metabolic risks may influence some aspects of postnatal human ontogeny, but do not account for prolongation of ontogeny independent of consideration of the developing human brain.

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